CASE REPORT

Radiofrequency ablation of a fascicular tachycardia after orthotopic cardiac transplantation

N Clarke, M Mason, V Paul

Abstract

A 51 year old male received an orthotopic transplant because of end stage ischaemic heart disease. The donor was a healthy male teenager with no history of arrhythmias or other cardiac conditions. The patient presented with haemodynamically stable tachycardia and dyspnoea five weeks post-transplant. The ECG showed a regular tachycardia of 140 beats/min with a right bundle branch block morphology, left axis deviation, and a QRS duration of 135 ms. There were independent P waves, capture, and fusion beats confirming the diagnosis of ventricular tachycardia. Endomyocardial biopsy showed moderate focal rejection that was thought to be responsible for the arrhythmia. During the following six months the patient had recurrent tachyarrhythmias; on each occasion the ECG morphology was the same and there was no cellular rejection. The patient continued to have frequent hospital admissions with ventricular tachycardia requiring DC cardioversion despite the empirical use of amiodarone, sotalol, disopyramide, and procainamide. Eighteen months after transplantation the diagnosis of fascicular tachycardia was suspected by ECG morphology and supported by successful termination with intravenous verapamil. The arrhythmia was successfully managed by radiofrequency ablation. This patient shows that arrhythmias following transplantation are not always related to rejection, and that other potentially reversible causes should be considered, particularly when the ECG during arrhythmia conforms to a classic configuration.

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The development of tachyarrhythmias in cardiac transplant patients is often taken as a possible indication of the development of acute rejection, but this is not necessarily the case. We describe a case of fascicular tachycardia after orthotopic cardiac transplantation and its successful management by radiofrequency ablation.

Case history

The patient was a 51 year old man who had received an orthotopic transplant because of end stage ischaemic heart disease. The donor was a healthy male teenager with no history of arrhythmias or other cardiac conditions and a normal resting electrocardiogram (ECG). The total ischaemic time of the donor heart was 4 hours and 40 minutes. Postoperative recovery was uneventful and the patient received routine immunosuppression with cyclosporin and azathioprine.

The baseline ECG during sinus rhythm had a right bundle branch block configuration and right axis deviation with a QRS duration of 135 ms (fig 1A).

The patient presented with haemodynamically stable tachycardia and dyspnoea five weeks post-transplant. The ECG showed a regular tachycardia of 140 beats/min with a right bundle branch block morphology, left axis deviation, and a QRS duration of 135 ms. There were independent P waves, capture, and fusion beats (fig 1B) confirming the diagnosis of ventricular tachycardia. His biochemical profile was normal and echocardiography confirmed good ventricular function with an ejection fraction of 70%.

The patient was electively cardioverted, and an endomyocardial biopsy showed moderate focal rejection (grade 3A) that was thought to be responsible for the arrhythmia. Immunosuppression was intensified with a three day pulse of intravenous methyprednisolone and horse antithymocyte globulin.

During the following six months the patient had recurrent tachyarrhythmias, including three episodes of documented ventricular tachycardia precipitated by endomyocardial biopsy. On each occasion the ECG morphology was the same and there was no cellular rejection. Investigation by ambulatory ECG recording, exercise testing, and coronary angiography did not reveal any abnormality. In addition, limited programmed stimulation studies failed to initiate the arrhythmia.

The patient continued to have frequent hospital admissions with ventricular tachycardia requiring DC cardioversion despite the empirical use of amiodarone, sotalol, disopyramide, and procainamide (alone and in combination).

Eighteen months after transplantation he was referred for further investigation. The

Department of Cardiology, Harefield Hospital, Harefield, Middlesex UB9 6JH, UK
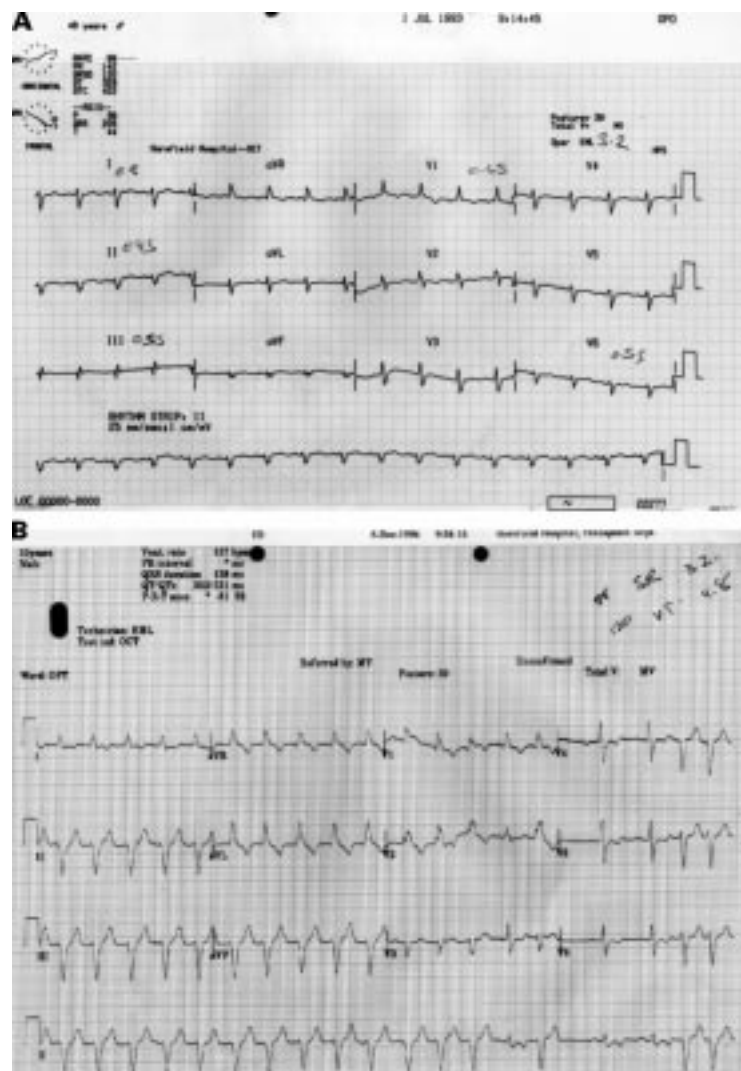N Clarke
M Mason
V Paul

Correspondence to:
Dr Paul.

Figure 1 (A) ECG during normal sinus rhythm. The numbers written by hand are the QRS deflections expressed in millivolts (summated QRS deflections are used as part of the routine surveillance for rejection in this centre). (B) ECG during tachycardia showing right bundle branch block, left axis deviation morphology. There is evidence of independent P wave activity (P) and a capture beat that breaks the tachycardia.
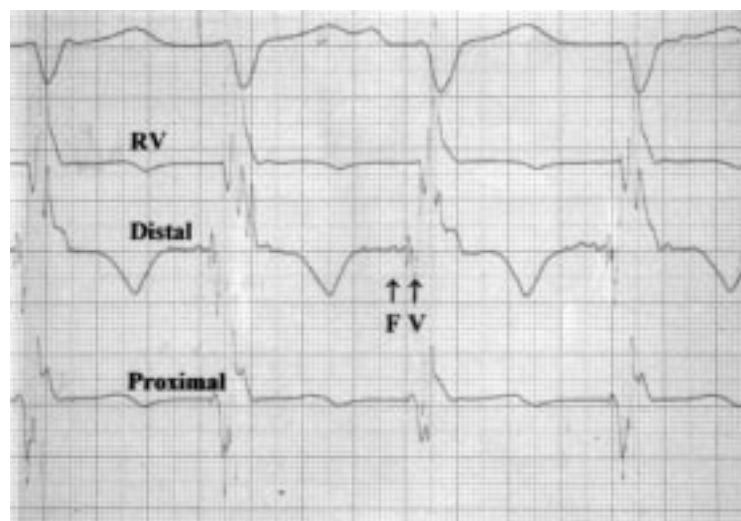

Figure 2 Surface ECG (top), right ventricular, distal, and proximal ablation catheter intracardiac electrograms showing the fascicular potential (F) as a high frequency discharge 20 ms before ventricular activation (V). (Paper speed 100 mm/s.)

diagnosis of fascicular tachycardia was suspected by ECG morphology and supported by successful termination with intravenous verapamil. Oral verapamil was not successful in preventing the arrhythmia.

Electrophysiological studies and radiofrequency ablation were done, during which the tachycardia was easily inducible. Fascicular tachycardia was confirmed by the demonstration of a reduced H–V interval during tachycardia and localisation of the focus to the left posterior septum.

At the successful ablation site, a fascicular potential was identified 40 ms before the onset of ventricular activation (fig 2). Two radiofrequency energy deliveries at 50 W were applied for 90 seconds. After energy delivery the tachycardia was no longer inducible.

The patient was discharged on no antiarrhythmic treatment and has remained free from the ventricular tachycardia for the past 24 months.

Discussion

Although ablation of an accessory pathway after transplantation has been described,[1] to our knowledge this is the first ablation of a fascicular tachycardia in an orthotopic cardiac transplant recipient.

Appropriate investigation and management of this patient had been delayed by failure to recognise the nature of the arrhythmia and by the belief that it had been precipitated by rejection. Although it has been suggested that there is coincident rejection in up to 70% of cases of atrial flutter,[2] the association with non-sustained ventricular tachycardia is less well established.[2 3] Furthermore, only the first episode of ventricular tachycardia was clearly associated with rejection in this patient.

The development of ventricular tachycardia with right bundle branch block morphology and left axis deviation in a young and otherwise normal heart suggests the diagnosis of fascicular tachycardia. The right bundle branch block seen on the resting ECG is a common finding in cardiac transplant patients and is of no prognostic significance.[4] The efficacy of verapamil and the potential for successful treatment with radiofrequency ablation in this type of ventricular tachycardia is well recognised.[5]

That the arrhythmia became a clinical problem soon after transplantation might be coincidental or a result of autonomic changes associated with transplantation.[6] As fascicular tachycardias may be sensitive to adrenergic stimulation,[5] the supersensitisation to circulating catecholamines, resulting from surgical denervation, may have played a role.

This patient serves as an example that the development of arrhythmias following transplantation is not always related to rejection, and that other potentially reversible causes should be considered, particularly when the ECG during arrhythmia conforms to a classic configuration.

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2 Scott CD, Dark JH, McComb JM. Arrhythmias after cardiac transplantation. *Am J Cardiol* 1992;**70**:1061–3.

3 Paul V, Mitchell A, O'Brien C, *et al*. The incidence and significance of conduction defects following orthotopic cardiac transplantation. *PACE* 1989;**12**:1215.

4 Gaita F, Giustetto C, LeClercq J-F, *et al*. Idiopathic verapamil responsive left ventricular tachycardia: clinical characteristics and long term follow up of 33 patients. *Eur Heart J* 1994;**15**:1252–60.

5 Babuty D, Aupart M, Cosnay P, *et al*. Electrocardiographic and electrophysiologic properties of cardiac allografts. *J Cardiovasc Electrophysiol* 1994;**5**:1053–63.

IMAGES IN CARDIOLOGY

Transoesophageal echocardiography diagnosis of a left coronary artery fistula draining into the right atrium

Most coronary artery fistulae are congenital, arising from incomplete obliteration of the primitive myocardial sinusoids and multiple fistulous tracts. Occasionally these fistulae may result from cardiac interventions such as percutaneous transluminal coronary angioplasty or endomyocardial biopsy. Involvement of the right coronary artery is marginally more common than the left, and drainage into the right atrium or ventricle is the rule, although drainage into the pulmonary artery, left heart, coronary sinus, and superior vena cava have been reported. Normally, the coronary artery feeding the fistula is dilated and runs a tortuous course, giving the serpiginous appearance illustrated in this case.

Patients with congenital coronary artery fistulae may be asymptomatic for many years; however, by middle age they often are symptomatic. Angina is the most common symptom because of coronary steal and coexisting atherosclerosis; however, some patients, as in this case, may present solely with exertional dyspnoea. Classically, a continuous murmur is described, but pansystolic and diastolic murmurs in isolation have been reported.

Although transthoracic echocardiography may be helpful in initial screening, a transoesophageal echocardiogram may be diagnostic and precise in delineating the fistulous vessel origin, course, and drainage. In addition, coexistent congenital cardiac disease, present in up to 20% of cases, can be confirmed or excluded. In the diagnosis of coronary artery fistulae, transoesophageal echocardiography is comparable to angiography, and may be superior.

(Top) Transoesophageal vertical plane image demonstrating the left atrium (LA) and right atrium (RA) with a tortuous left coronary artery fistula (F) draining into the right atrium. (Bottom) Same as above but demonstrating flow from the fistula into the right atrium.

P R JAMES
D DAWSON
S M C HARDMAN